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=> s nephrotoxicity or neuropathy or proteinuria or glomerular(w)filtration(w)rate
L1      349451 NEPHROTOXICITY OR NEUROPATHY OR PROTEINURIA OR GLOMERULAR(W)
               FILTRATION(W) RATE

=> s estradiol(w)metabolite or 2-hydroxyestradiol or 2-methoxyestradiol or
4-hydroxyestradiol or 4-methoxyestradiol
L2        4559 ESTRADIOL(W) METABOLITE OR 2-HYDROXYESTRADIOL OR 2-METHOXYESTRAD
               IOL OR 4-HYDROXYESTRADIOL OR 4-METHOXYESTRADIOL

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L3          51 L1 AND L2

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                 ANSWERS '13-45' FROM FILE EMBASE

=> d ti au abs so py 1-8

L4   ANSWER 1 OF 45  CAPLUS  COPYRIGHT 2006 ACS on STN DUPLICATE 1
TI   Estradiol Metabolites Attenuate Renal and
     Cardiovascular Injury Induced by Chronic Nitric Oxide Synthase Inhibition
AU   Tofovic, Stevan P.; Salah, Eman M.; Dubey, Raghvendra K.; Melhem, Mona F.;
     Jackson, Edwin K.
AB   Our previous studies in rodent models of nephropathy demonstrate that
     2-hydroxyestradiol (2HE), an estradiol
     metabolite with little estrogenic activity, exerts reno-protective
     effects.  In vivo, 2HE is readily converted to 2-
     methoxyestradiol (2ME), a major estradiol
     metabolite with no estrogenic activity.  This study was to determine
     whether 2ME has renal and cardiovascular protective effects in vivo.
     First, the acute (90 min) and chronic (14 days) effects of 2ME (10
     μg/kg/h) on blood pressure and renal function were examined in
     normotensive and spontaneously hypertensive rats (SHR).  Second, a rat
     model of cardiovascular and renal injury induced by chronic nitric oxide
     synthase inhibition (Nω-nitro-L-arginine; 40 mg/kg/d; LNNA group)
     was used to examine the protective effects of estradiol
     metabolites.  Subsets of LNNA-treated rats were administered
     either 2HE or 2ME (10 μg/kg/h via osmotic minipump); LNNA+2ME and
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LNNA+2HE groups, resp. 2-Methoxyestradiol had no acute or chronic effects on blood pressure or renal function in normotensive animals or on hypertension in SHR. Prolonged, 5-wk NOS inhibition induced severe cardiovascular and renal disease and high mortality (75%, LNNA group). 2ME, but not 2HE, significantly decreased elevated blood pressure and attenuated the reduction in GFR. 2HE delayed the onset of proteinuria, whereas no proteinuria was detected in the 2-ME group. 2HE and 2ME reduced mortality rate by 66% and 83%, resp. ($P < 0.001$). In the kidney, 2HE and 2ME abolished LNNA-induced interstitial and glomerular inflammation, attenuated glomerular collagen IV synthesis, and inhibited glomerular and tubular cell proliferation. In the heart, 2HE and 2ME markedly reduced vascular and interstitial inflammation and reduced collagen synthesis and vascular/interstitial cell proliferation. Thus, in a model of severe cardiovascular and renal injury, 2-methoxyestradiol (a major nonestrogenic estradiol metabolite) exerts renal and cardiovascular protective effects and reduces mortality.

SO Journal of Cardiovascular Pharmacology (2005), 46(1), 25-35
CODEN: JCPCDT; ISSN: 0160-2446
PY 2005

L4 ANSWER 2 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2

TI 2-Hydroxyestradiol Attenuates Renal Disease in Chronic Puromycin Aminonucleoside Nephropathy

AU Tofovic, Stevan P.; Dubey, Raghvendra; Salah, Eman M.; Jackson, Edwin K.

AB It has been previously shown that 2-hydroxyestradiol (2-OHE) attenuates the development of renal disease in genetic nephropathy associated with obesity and the metabolic syndrome. The purpose of this study was to test the hypothesis that 2-OHE, irresp. of its effects on metabolic status and/or obesity, exerts direct renoprotective effects in rats in vivo. First, the effects of increasing doses of 2-OHE on mesangial cell growth, proliferation, and collagen synthesis in isolated rat glomerular mesangial cells were evaluated in vitro. Second, the effects of 12-wk administration of 2-OHE (10 μg/h/kg) on renal function and structure in chronic puromycin aminonucleoside (PAN)-induced nephropathy in rats were evaluated in vivo. 2-OHE concentration-dependently (0.001 to 1 M) inhibited serum (2.5%)-induced cell growth (3H-thymidine incorporation), collagen synthesis (3H-proline incorporation), and cell proliferation (cell number). Importantly, the inhibitory effects of 2-OHE (0.1 μM) were not blocked by ICI182780 (50 μM), an estrogen receptor antagonist. In vivo, chronic administration of PAN (75 mg/kg + 5 + 20 mg/kg) over 12 wk induced severe chronic renal disease. Chronic treatment with 2-OHE significantly attenuated PAN-induced decrease in glomerular filtration, reduced proteinuria, and the elevated BP, and it had no effect on PAN-induced increase in plasma cholesterol and triglycerides levels. 2-OHE had no effects on plasma testosterone levels in male nephropathic animals. Immunohistochem. staining for collagen IV and proliferating cell nuclear antigen (PCNA) in glomeruli and transforming growth factor-β (TGF-β) in renal tubular cells were significantly higher in PAN nephropatic rats vs. control animals with intact kidneys. PAN also markedly increased glomerular and interstitial macrophage infiltration (ED1+ cells). 2-OHE had no effects on renal tubular cell TGF-β, but it significantly reduced glomerular PCNA and collagen IV and glomerular and interstitial macrophage infiltration. In summary, this study provides the first evidence that 2-OHE exerts direct renoprotective effects in vivo. These effects are mediated by estrogen receptor-independent mechanisms and are due, at least in part, to the inhibition of some of the key proliferative mechanisms involved in glomerular remodeling and sclerosis.

SO Journal of the American Society of Nephrology (2002), 13(11), 2737-2747
CODEN: JASNEU; ISSN: 1046-6673
PY 2002

L4 ANSWER 3 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN

TI Long acting injectable crystal formulations of estradiol metabolites and methods of using same
IN Allison, S. Dean
AB The present invention provides sustained release formulations of estradiol metabolites whereby the in vivo pharmacokinetics are manipulated by a method selected from the group consisting of chemical modification, crystal packing formation, particle size or a combination thereof. Such compns. are useful in the long-term treatment of a wide variety of diseases. For example, a 4% weight/volume solution of 2-methoxyestradiol (2ME) was prepared in a solvent consisting of 9.2% THF, 61% methanol and 28% aqueous 6 M hydrochloric acid. This solution was added dropwise to an equal volume of vigorously stirring water. The resulting solid was isolated by suction filtration, washed with water, and dried under vacuum. The resulting 2ME crystals were a mixture of large, hollow prisms greater than 500 μm in length by approx. 200 μm width, and cubic particles, ranging from approx. 50 μm down to approx. 500 nm square. The broad particle size range was intended to give a complex, biphasic pharmacokinetic profile upon injection.
SO PCT Int. Appl., 27 pp.
CODEN: PIXXD2
PY 2006

L4 ANSWER 4 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
TI Administration of estradiol metabolites for the treatment of drug-induced nephrotoxicity and related conditions
IN Tofovic, Stevan P.; Dubey, Raghvendra K.; Jackson, Edwin K.
AB Methods and composition are provided for the treatment or prevention of drug-induced nephrotoxicity and related conditions. More particularly, this invention relates to compns. comprising estradiol metabolites, and prodrugs thereof, that may be incorporated in various controlled release formulations.
SO PCT Int. Appl., 30 pp.
CODEN: PIXXD2
PY 2004
2004
2004
2004

L4 ANSWER 5 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
TI Administration of estradiol metabolites for the treatment or prevention of obesity, metabolic syndrome, diabetes, and vascular and renal disorders
IN Jackson, Edwin K.; Tofovic, Stevan P.; Dubey, Raghvendra K.
AB Methods are provided for preventing or treating risk factors for cardiovascular disease in an individual, comprising administering a therapeutically effective amount of a composition comprising an estradiol metabolite to said individual. Such risk factors include obesity, the metabolic syndrome, diabetes mellitus, vascular disorders, and renal disorders. Preferred estradiol metabolites include 2-methoxyestradiol, 4-methoxyestradiol, 2-hydroxyestradiol, and 4-hydroxyestradiol or prodrugs thereof. The compns. may also be in the form of a controlled release formulation. Methods are also provided for use of estradiol metabolites to treat or prevent insulin resistance, vascular endothelial dysfunction, hyperlipidemia, hypertension, diabetic nephropathy, proteinuria and reducing leptin levels. In addition, the methods provide a method of stabilizing glucose levels. These treatments may be used in either gender because of their lack of a feminizing estrogenic effect.
SO PCT Int. Appl., 51 pp.
CODEN: PIXXD2
PY 2003
2004

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L4 ANSWER 6 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
TI Monofunctional and/or polyfunctional polylysine conjugates for treatment of neural disorders, autoimmune diseases, and proliferative diseases
IN Geffard, Michel
AB The use of polylysine for preparing pharmaceutical compns. or combinations useful for treating neural degeneration, infectious, traumatic and toxic neuropathies, auto-immune degenerative diseases and proliferative diseases, is disclosed. Polylysine conjugates are also disclosed.
SO PCT Int. Appl., 44 pp.
CODEN: PIXXD2
PY 1996
1996
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L4 ANSWER 7 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Administration of estradiol metabolites for the treatment or prevention of obesity, metabolic syndrome, diabetes, and vascular and renal disorders.
AU Jackson, Edwin K. [Inventor]; Tofovic, Stevan P. [Inventor]; Dubey, Raghvendra K. [Inventor]
AB Methods are provided for preventing or treating risk factors for cardiovascular disease in an individual, comprising administering a therapeutically effective amount of a composition comprising an estradiol metabolite to said individual. Such risk factors include obesity, the metabolic syndrome, diabetes mellitus, vascular disorders, and renal disorders. Preferred estradiol metabolites include 2-methoxyestradiol, 4-methoxyestradiol, 2-hydroxyestradiol , and 4-hydroxyestradiol or prodrugs thereof. The compositions may also be in the form of a controlled release formulation. Methods are also provided for use of estradiol metabolites to treat or prevent insulin resistance, vascular endothelial dysfunction, hyperlipidemia, hypertension, diabetic nephropathy, proteinuria and reducing leptin levels. In addition, the methods provide a method of stabilizing glucose levels. These treatments may be used in either gender because of their lack of a feminizing estrogenic effect.
SO Official Gazette of the United States Patent and Trademark Office Patents, (FEB 14 2006)
CODEN: OGUPE7. ISSN: 0098-1133.
PY 2006

L4 ANSWER 8 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Estradiol metabolites attenuate renal and cardiovascular injury induced by chronic angiotensin II administration.
AU Tofovic, Stevan P. [Reprint Author]; Mady, Hussam; Jackson, Edwin K. [Reprint Author]

SO Journal of the American Society of Nephrology, (November 2003) Vol. 14, No. Abstracts Issue, pp. 620A. print.
Meeting Info.: Meeting of the American Society of Nephrology Renal Week. San Diego, CA, USA. November 12-17, 2003. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
PY 2003

=> d ti au abs so py 8-20

L4 ANSWER 8 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Estradiol metabolites attenuate renal and cardiovascular injury induced by chronic angiotensin II administration.
AU Tofovic, Stevan P. [Reprint Author]; Mady, Hussam; Jackson, Edwin K. [Reprint Author]
SO Journal of the American Society of Nephrology, (November 2003) Vol. 14, No. Abstracts Issue, pp. 620A. print.
Meeting Info.: Meeting of the American Society of Nephrology Renal Week. San Diego, CA, USA. November 12-17, 2003. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
PY 2003

L4 ANSWER 9 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Renoprotective effects of 2-methoxyestradiol in streptozotocin diabetic rats.
AU Tofovic, Stevan P. [Reprint Author]; Dubey, Raghvendra [Reprint Author]; Jackson, Edwin K. [Reprint Author]
SO Journal of the American Society of Nephrology, (November 2003) Vol. 14, No. Abstracts Issue, pp. 126A. print.
Meeting Info.: Meeting of the American Society of Nephrology Renal Week. San Diego, CA, USA. November 12-17, 2003. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
PY 2003

L4 ANSWER 10 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI The new angiotherapy.
AU Fan, Tai-Ping D. [Reprint author]; Kohn, Elise C.
AB Angiogenesis is a feature in several pathological conditions, including cancer, atherosclerosis, rheumatoid arthritis, and diabetic neuropathy. In this book, an international team of researchers review the recent progress in basic and applied angiogenesis research. The book covers key concepts in the physiology and pathophysiology of angiogenesis, evaluates the potential of angiotherapy in the management of angiogenic disease, discusses angiogenic and antiangiogenic agents in development and in clinical trials, and considers the potential of gene therapy in the development of angiotherapy. There are six major sections in this book that include 30 individually authored chapters. This book is useful for those who want to understand the central role of human angiogenesis, as well as the new therapeutic methods that are now possible because the ability to control vascular development has been achieved.
SO Fan, Tai-Ping D. [Editor]; Kohn, Elise C. [Editor]. (2002) pp. i-xix, 1-609. The new angiotherapy. print.
Publisher: Humana Press Inc., 999 Riverview Drive, Suite 208, Totowa, NJ, 07512, USA.
ISBN: 0-89603-464-X (cloth).
PY 2002

L4 ANSWER 11 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Estradiol metabolites attenuate renal injury induced by chronic nitric oxide synthase inhibition.
AU Tofovic, Stevan P. [Reprint author]; Dubey, Raghvendra [Reprint author]; Jackson, Edwin K. [Reprint author]

SO Journal of the American Society of Nephrology, (September, 2002) Vol. 13, No. Program and Abstracts Issue, pp. 156A. print.
Meeting Info.: Meeting of the American Society of Nephrology. Philadelphia, PA, USA. October 30-November 04, 2002. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
PY 2002

L4 ANSWER 12 OF 45 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Renoprotective effects of 2-hydroxyestradiol.
AU Tofovic, Stevan P. [Reprint author]; Dubey, Raghvendra K. [Reprint author]; Bastacky, Sheldon I.; Jackson, Edwin K. [Reprint author]
SO Journal of the American Society of Nephrology, (September, 2001) Vol. 12, No. Program and Abstract Issue, pp. 86A. print.
Meeting Info.: ASN (American Society of Nephrology)/ISN (International Society of Nephrology) World Congress of Nephrology. San Francisco, CA, USA. October 10-17, 2001.
CODEN: JASNEU. ISSN: 1046-6673.
PY 2001

L4 ANSWER 13 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
TI Epothilones in the treatment of cancer.
AU Larkin J.M.G.; Kaye S.B.
AB Epothilones are cytotoxic macrolides with a similar mechanism of action to paclitaxel but with the potential advantage of activity in taxane-resistant settings in preclinical models. The epothilones ixabepilone, patupilone, BMS-310705, KOS-862 and ZK-EPO are in early clinical trials for cancer treatment. Phase I studies have shown that dose-limiting toxicities of epothilones are generally neurotoxicity and neutropoenia although initial studies with patupilone indicated that diarrhoea was dose limiting. Neuropathy induced by ixabepilone may be schedule dependent. Over 20 Phase II studies of epothilones in cancer treatment have been reported, and significant activity in taxane-sensitive tumour types (such as breast, lung and prostate cancers) has been noted. Response rates in taxane-refractory metastatic breast cancer are relatively modest, but ixabepilone and patupilone have shown promising efficacy in hormone-refractory metastatic prostate cancer and in taxane-refractory ovarian cancer. .COPYRGT. 2006 Informa UK Ltd.
SO Expert Opinion on Investigational Drugs, (2006) Vol. 15, No. 6, pp. 691-702. .
Refs: 100
ISSN: 1354-3784 CODEN: EOIDER
PY 2006

L4 ANSWER 14 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
TI Antitubulin agents for the treatment of cancer - A medicinal chemistry update.
AU Mahindroo N.; Liou J.-P.; Chang J.-Y.; Hsieh H.-P.
AB The antitubulin agents taxanes and Vinca alkaloids form the first-line of treatment in clinical oncology for many cancers. The crucial role of microtubules in cell division has made antitubulin agents the focus of research, with sustained efforts to find new agents and to improve the profile of known agents by overcoming multi-drug resistance (MDR) and improving the druggability. The present review updates the medicinal chemistry of antitubulin agents covering the patents and literature published from May 2002 to November 2005. The antitubulin agents have been broadly classified into microtubule-destabilising agents, microtubule-stabilising agents and kinesin-like spindle protein inhibitors. This review provides an insight into the diversity of the chemical classes with antitubulin mechanisms of anticancer activity. .COPYRGT. 2006 Informa UK Ltd.

SO Expert Opinion on Therapeutic Patents, (2006) Vol. 16, No. 5, pp. 647-691.
.
Refs: 168
ISSN: 1354-3776 CODEN: EOTPEG
PY 2006

L4 ANSWER 15 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
TI Treatment of multiple myeloma: An emphasis on new developments.
AU Kyle R.A.; Rajkumar S.V.
AB Not all patients who fulfill the minimal criteria for diagnosis of multiple myeloma should be treated. If doubt exists about beginning therapy, one should wait and re-evaluate the patient in 2 or 3 months. There is no evidence that early treatment of multiple myeloma is advantageous. All patients should be considered possible candidates for an autologous stem cell transplantation. If they are deemed to be eligible, they should be treated for 3 to 4 months with therapy that does not damage the hematopoietic stem cells. Currently, most physicians use thalidomide plus dexamethasone or dexamethasone alone for induction. Vincristine, doxorubicin (Adriamycin), and dexamethasone (VAD) have been used in the past. Autologous stem cell transplantation prolongs disease-free survival and overall survival. The treatment-related mortality rate is 1% to 2%. Melphalan, 200 mg/m(2), is the most widely used preparative regimen. Although allogeneic transplantation is attractive, the mortality rate (about 20%) is too high to recommend conventional allogeneic transplantation. Non-myeloablative transplantation is currently under investigation. If the patient is not a candidate for autologous stem cell transplantation, therapy with melphalan and prednisone is a good choice. Patients with relapsed or refractory disease may be treated with dexamethasone, thalidomide and dexamethasone, bortezomib (Velcade, PS-341), or lenalidomide (Revlimid, not yet approved by the Food and Drug Administration). .COPYRGT. 2006 Taylor & Francis.
SO Annals of Medicine, (2006) Vol. 38, No. 2, pp. 111-115. .
Refs: 23
ISSN: 0785-3890 E-ISSN: 1651-2219 CODEN: ANMDEU
PY 2006

L4 ANSWER 16 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
TI Current status and perspective of antiangiogenic therapy for cancer: Urinary cancer.
AU Kanda S.; Miyata Y.; Kanetake H.
AB Angiogenesis is considered a prerequisite for solid tumor growth. Antiangiogenic therapy reduces tumor size and extends host survival in a number of preclinical animal models. However, in humans antiangiogenic therapy is a poor promoter of tumor regression and has shown minimal effect on patient survival. In urinary cancers, such as renal cell cancer, prostate cancer, and bladder cancer, advanced refractory disease is a good candidate for antiangiogenic therapy because of its resistance to ordinary chemotherapy, radiotherapy, and hormonal therapy. Unique characteristics of molecular mechanisms underlie the induction of angiogenesis in urinary cancers. In this review, we summarize these unique mechanisms and review the results of clinical trials of antiangiogenic therapy for these cancers, discussing prospects and problems relating to antiangiogenic therapy. .COPYRGT. The Japan Society of Clinical Oncology 2006.
SO International Journal of Clinical Oncology, (2006) Vol. 11, No. 2, pp. 90-107. .
Refs: 268
ISSN: 1341-9625 CODEN: IJCOF6
PY 2006

L4 ANSWER 17 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Phase I clinical trial of oral 2-methoxyestradiol, an antiangiogenic and apoptotic agent, in patients with solid tumors.
AU Dahut W.L.; Lakhani N.J.; Gulley J.L.; Arlen P.M.; Kohn E.C.; Kotz H.; McNally D.; Pair A.; Nguyen D.; Yang S.X.; Steinberg S.M.; Venitz J.; Sparreboom A.; Figg W.D.
AB Purpose: To determine the maximum-tolerated dose (MTD) and toxicity profile of the novel anticancer agent, 2-methoxyestradiol (2ME2) administered orally, in patients with solid tumors. Materials and methods: Twenty patients with refractory solid tumors were enrolled. 2ME2 was given orally starting at 400 mg bid with dose escalation until 3000 mg bid. Tumor biopsies were taken before and after starting the drug to assess for microvessel density by CD 31 and cell proliferation by Ki67 immunohistochemistry. Serial plasma samples collected up to 50 hours after first single oral dose for characterization of pharmacokinetics, were analyzed using liquid chromatography tandem mass-spectrometry. Results: Eleven men and nine women received 2ME2 at dose levels of 400 mg bid (n = 3), 800 mg bid (n = 3), 1600 mg bid (n = 6), 2200 mg bid (n = 5) and 3000 mg bid (n = 3). There were no dose limiting toxicities, therefore the MTD was not defined. There was one episode of grade 4 angioedema in the 1600 mg bid dose level 38 days into 2ME2 treatment. Other toxicities were mild to moderate. A patient with clear cell carcinoma of the ovary had a partial response at 1600 mg bid dose level lasting over three years. Conclusion: MTD for 2ME2 was not reached at dose of 3000 mg bid. The trial was closed due to extremely low plasma concentrations of 2ME2 relative to the doses administered. 2ME2 treatment had no effect on microvessel density (CD31 immunostaining) and cell proliferation (Ki-67 immunostaining). A new formulation of 2ME2 with improved bioavailability is currently being developed. .COPYRGT.2005 Landes Bioscience.
SO Cancer Biology and Therapy, (2006) Vol. 5, No. 1, pp. 22-27. .
Refs: 32
ISSN: 1538-4047 E-ISSN: 1555-8576
PY 2006

L4 ANSWER 18 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
TI Use of angiogenesis inhibitors in tumour treatment.
AU Fayette J.; Soria J.-C.; Armand J.-P.
AB Advances in molecular biology have permitted the characterisation of mechanisms underlying angiogenesis. Angiogenesis is a crucial process in tumour pathogenesis as it sustains malignant cells with nutrients and oxygen. It is well known that tumour cells secrete various growth factors including VEGF, which triggers endothelial cells to form new capillaries. Preventing the expansion of new blood vessel networks results in reduced tumour size and metastases. Not surprisingly, numerous drugs that are currently under clinical development interfere with growth factor-derived angiogenic signals. This review aims to describe angiogenesis inhibitors and surveys their different modes of action. .COPYRGT. 2005 Elsevier Ltd. All rights reserved.
SO European Journal of Cancer, (2005) Vol. 41, No. 8, pp. 1109-1116. .
Refs: 53
ISSN: 0959-8049 CODEN: EJCAEL
PY 2005

L4 ANSWER 19 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
TI Fifth International Prostate Cancer Congress. 1-3 July 2005, Rio Grande, Puerto Rico.
AU Slovin S.F.
AB This is an extraordinary time for the treatment of localized and metastatic prostate cancer as manifested by the multi-disciplinary and multi-modality approaches being incorporated into standard technologies. While this is merely a highlight of the current state-of-the-art processes, and does not reflect the importance of any one approach as

being superior to the next, the information shared at this meeting will continue to provide a platform, which will establish more diverse treatments. This will not only impact on the disease itself, but may help delay time-to-radiographic progression and improve overall survival. .COPYRGT. The Thomson Corporation.

SO IDrugs, (2005) Vol. 8, No. 9, pp. 710-712. .
ISSN: 1369-7056 CODEN: IDRUFN

PY 2005

L4 ANSWER 20 OF 45 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Angiogenesis: A curse or cure?.

AU Gupta K.; Zhang J.

AB Angiogenesis, the growth of new blood vessels is essential during fetal development, female reproductive cycle, and tissue repair. In contrast, uncontrolled angiogenesis promotes the neoplastic disease and retinopathies, while inadequate angiogenesis can lead to coronary artery disease. A balance between pro-angiogenic and antiangiogenic growth factors and cytokines tightly controls angiogenesis. Considerable progress has been made in identifying these molecular components to develop angiogenesis based treatments. One of the most specific and critical regulators of angiogenesis is vascular endothelial growth factor (VEGF), which regulates endothelial proliferation, permeability, and survival. Several VEGF based treatments including anti-VEGF and anti-VEGF receptor antibodies/agents are in clinical trials along with several other antiangiogenic treatments. While bevacizumab (anti-VEGF antibody) has been approved for clinical use in colorectal cancer, the side effects of antiangiogenic treatment still remain a challenge. The pros and cons of angiogenesis based treatment are discussed.

SO Postgraduate Medical Journal, (2005) Vol. 81, No. 954, pp. 236-242. .
Refs: 61
ISSN: 0032-5473 CODEN: PGMJAO

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L2 4559 S ESTRADIOL(W)METABOLITE OR 2-HYDROXYESTRADIOL OR 2-METHOXYESTR
L3 51 S L1 AND L2
L4 45 DUPLICATE REMOVE L3 (6 DUPLICATES REMOVED)

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